NON-ARTICULAR RHEUMATISM*

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A PHYSICIAN who works in a clinic or research unit devoted to the problems of rheumatic diseases cannot help but be impressed with the high incidence and the large variety of rheumatic disorders involving non-articular connective tissue structures. Collectively, this group of clinical entities has been designated *non-articular rheumatism.* It has been the author's experience that approximately one of every 4 patients referred to the physician or clinic specializing in rheumatic diseases, with a diagnosis of "arthritis," does not have joint disease; instead he has a form of non-articular rheumatism. Confusion in the mind of the physician or the patient is not surprising for the major symptoms of each group of disorders are similar, namely, pain and tenderness in region of joints, and stiffness and limitation of movement of joints. But in the non-articular disorders the joints are normal; the symptoms arise from abnormalities affecting chiefly the structures which move them; the joint movement initiates or aggravates the symptoms and for this reason the inexperienced person concludes, erroneously, that the joint itself is diseased; that the patient has arthritis. It is very important that forms of non-articular rheumatism be differentiated from joint disease, for two reasons: the importance to the physician in managing the patient, for appropriate treatment of the two groups differs; and for the patient's sake, the prognosis and significance of the disease to the patient is vastly different depending upon whether or not joints are affected.

The non-articular rheumatic illnesses include a large number of clinical entities (Table I). It must be appreciated that the designation of non-articular rheumatism applies only to those conditions affecting the non-articular connective tissue directly; it does not include the febrile illnesses, particularly infectious diseases in which rheumatic symptoms may be prominent in the prodromal or early clinical stages. Sometimes in illnesses, such as influenza, the patient's first chief complaint

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TABLE I. CLINICAL FORMS OF NON-ARTICULAR RHEUMATISM

Fibrositis, generalized	Periarthritis (para-arthritis)
Fibrositis, localized:	Panniculitis
Painful stiff neck	Herniated subcutaneous fat
"Lumbago"	Reflex neuromuscular dystrophy
Bursitis	"shoulder-hand syndrome"
Tenosynovitis	"Psychogenic rheumatism"
Tendonitis	(rheumatic manifestations of psycho-
"Tendon attachment syndrome"	neurosis)
Fasciitis	

may be back-ache or generalized aching; however, as the disease progresses the rheumatic symptoms lessen or disappear and the clinical problem is recognized to be non-rheumatic.

FIBROSITIS

The commonest form of non-articular rheumatism affects the fibrous connective tissue and consequently has been called "fibrositis." Usually the nature of the fibrous tissue pathology appears not to be inflammatory, hence the term "fibrositis" is incorrect if strictly interpreted. Even so "fibrositis" is the designation commonly used, and this term is employed in the discussion in the American Rheumatism Association Primer.[1]

Non-articular fibrous connective tissue always shares, to a varying degree, in the abnormalities which exist in patients who have rheumatoid arthritis; sometimes the fibrositis is the predominant feature of this illness. Similarly, affection of the connective tissue around osteoarthritic joints accounts for much of the symptomatology of this form of arthritis. Because the chief pathology in these forms of arthritis is in the joints, the non-articular fibrous tissue disorder associated therewith is called "*secondary* fibrositis," or the fibrositis of rheumatoid or osteoarthritis. But the same disorder of non-articular connective tissue frequently occurs without disease of the joints; this clinical entity is called "*primary* fibrositis." Other names for this entity include "myofibrositis," "myalgia" and "muscular rheumatism," the latter term being quite common among the laity. It is this condition, primary fibrositis, to which attention is now directed.

Clinical Manifestations: In order to establish this entity clearly, for there has been much confusion concerning it, the clinical features will

be reviewed first. Primary fibrositis may exist simultaneously and widely scattered in many parts of the body, i.e., generalized fibrositis; or it may be sharply localized. The more disseminated form involves the fascia, tendons, aponeuroses particularly in the back, shoulders, thighs and hands, although all other fibrous tissue may be affected. The clinical pattern of primary fibrositis is quite consistent. Affected persons are usually young or middle-aged adults. The illness may begin acutely and cause very severe pain, but when it is more generalized, the onset is usually insidious and the development gradual so that the disease is commonly a chronic disorder. Aching and stiffness of the neck, the interscapular upper back, the lumbosacral region or the entire back, shoulders and thighs are common early symptoms. Turning, stooping and twisting motions of the back, abduction and rotation at the shoulders and lying on the side are difficult to do and aggravate the symptoms. Often the hands become stiff; movement of the fingers is difficult; the hands feel clumsy, and grasp becomes so weak that objects are dropped and work with the hands becomes difficult or impossible. The patients tend to tire easily and, because of the discomfort and sense of fatigue, they avoid more strenuous physical activities and may follow a schedule of self-imposed, partial invalidism.

An important feature of this syndrome is the rather consistent effect of certain factors upon the symptoms. Pain and stiffness are usually worse after prolonged rest, consequently patients are most uncomfortable in early morning after a night's sleep. Ordinary activities or planned exercises of the affected parts almost invariably limber the patient and relieve aching. Patients feel best in mid-day; at the end of the day fatigue adds to the symptoms; while sitting in the theatre, at cards or at a desk there is usually no discomfort but with activity thereafter there is considerable aching and stiffness. The stiffness which develops in muscles and other connective tissues with rest and in the cold is much like the "jelling" that occurs in gelatine mixtures when they are refrigerated. Dampness, cold, rain and snow usually worsen these phenomena; warmth, dryness and high barometric pressure characteristically relieve, consequently many persons who have fibrositis suffer more in winter and spring and feel better in the summer and early fall.

Physical signs of this disease syndrome are few. Inspection and palpation reveal no connective tissue abnormality except for mild atrophy of muscle when joint motion has been impaired for a long period. There

are never signs of inflammation or other abnormalities at any joints but due to pain and stiffness there may be limitation of movement of some joints. There may be little or no tenderness but at sites of greater trouble there may be moderate, generalized tenderness. Sometimes great tenderness exists at sharply localized regions from which pain radiates through the distribution of fibrous tissue structure. These have been called "trigger points" or "trigger zones" of pain, and "myalgic spots." Earlier writers described "fibrositic nodules," as almond-sized, tender masses of firm, rubbery consistency commonly located in the lower lumbar and sacral region. However, such nodules are often encountered in non-rheumatic individuals, and are considered by most current investigators not to be part of the fibrositic syndrome.

The patient with fibrositis is in good systemic health and does not appear ill. Fever and loss of weight do not occur. The syndrome usually continues for many months or years, with symptoms fluctuating through an undulating course. Frequently there are remissions and recurrences. Seldom is the disease sufficiently severe or persistent to incapacitate the patient. Truly, it is more of a "nuisance" disorder.

Laboratory Observations: Results of usual laboratory tests are characteristically normal. There is no anemia or leukocytosis; the erythrocyte sedimentation rate is normal or only slightly elevated and roentgenograms of joints show no changes in cartilages or bones. Creatinuria has been reported to be somewhat proportional to the symptoms.[2] Electromyographic studies have shown increased irritability of muscles in some cases, but no abnormality in most.[2,3]

Pathology: Early reports contain descriptions of tissue in biopsies of muscle and "fibrositic nodules." Muscle and white fibrous tissue have been described as showing varying amounts of degeneration and atrophy of the fibres, and sometimes small collections of cellular exudate about some of the more abnormal fibres. The "nodules" were described as collections of edematous fibrous tissue, usually in loose, fatty, subcutaneous tissue. Recently many workers have reported studies of the pathology of muscle and other connective tissue as revealing no characteristic tissue change and they consider, instead of their being an anatomic tissue change, that there are chemical or physio-chemical changes in the connective tissue, possibly due to enzymatic disturbances which alter the physiology of the tissues involved.

Etiology: The cause of primary fibrositis is unknown. The same

uncertainties surround the nature of this rheumatic disorder as exist for rheumatoid arthritis. Sometimes infection, trauma, unusual physical activity, fatigue, dampness and cold appear to be "trigger mechanisms" or contributory causes. In most instances the illness seems to originate "out of a clear sky." Inherited fibrous tissue characteristics may set the background for this ailment.

The lack of demonstrable, characteristic, microscopic pathology in the fibrous tissue, the mystery surrounding the etiology, and the different locations of disturbances sometimes included under the category "fibrositis," have made a few conservative rheumatologists object to the concept of fibrositis. Some physicians insist that there is no such disorder and condemn the use of the term "fibrositis," and they ridicule this diagnosis, as well as the physicians who make it. But I direct your attention to the very consistent, clinical pattern of the rheumatic symptoms and dysfunction which characterize fibrositis unassociated with any demonstrable joint disease, and unaccompanied by indications of any inflammatory process or systemic illness. This syndrome differs so much from other forms of rheumatism that it certainly should be differentiated and managed as a distinct entity. *It is this clinical syndrome*, not a cellular pathologic process producing anatomic damages, that makes up the form of rheumatism labeled "fibrositis." Although this term is admittedly poor (its connotation is ambiguous), it serves to designate a distinctly different clinical entity just as well as the terms "rheumatoid," and "osteoarthritis" which, when examined critically are found to be poor terms for the diseases they label, but which terms common usage has long since made acceptable.

The diagnosis "fibrositis" should be restricted to the clinical disorder described. Simply because the etiology and pathologic features of this syndrome are ill-defined, it is not justifiable to diagnose every rheumatic process free of demonstrable joint pathology as "fibrositis." This has been done too often and presents another reason for some physicians objecting to considering fibrositis as being a distinct clinical syndrome.

Difference in conception of fibrositis is noted in the reports concerning the relative incidence of different rheumatic diseases (Table II). The incidence of "fibrositis" is reported much greater in British civilian and military personnel than among Americans. Although Great Britain is the "home of fibrositis," this difference is more likely due to the fact that other forms of non-articular rheumatism were not separated from

TABLE II. RELATIVE INCIDENCE OF "FIBROSITIS" REPORTED IN RECENT YEARS

Author	*Country*	*Status*	*Per Cent*
Fletcher and Lewis-Taning	England	Civilian	45
Schmidt	England	Civilian — miners	42
Boland	U. S.	Army General Hosp.	5
Boland and Corr	U. S.	Army General Hosp.	7
Savage	Gr. Britain	E.T.O. Field Army	52
		(General Hospital)	20
	Scotland	Army depot	70
Copeman	British	Military	70
Kersley	British	Military — Far East	33

this category. In the United States "psychogenic rheumatism" (rheumatic manifestations of psychoneurosis) is separately classified whereas in England it is not. This undoubtedly lowers the frequency of fibrositis statistics from the United States and further emphasizes the wisdom of restriction of use of the designation fibrositis to a definite syndrome.

Diagnosis: A characteristic clinical history, absence of pathologic joint changes, existence of normal systemic health and negative laboratory findings are the basis for the diagnosis of fibrositis. Differentiation from classical rheumatoid or osteoarthritis is not difficult except in early stages when, in some instances, it may be necessary to study the condition carefully and observe the course of the illness for several months before differentiation is clear. Features distinguishing fibrositis from psychogenic rheumatism will be presented hereafter.

Management: Treatment of fibrositis is supportive and symptomatic.

It is usually helpful for the patient to avoid strenuous physical activity and to procure additional rest during the daytime, but it is a mistake to curtail ordinary activity and recreation that is not fatigue-producing. Mild exercise especially after application of heat usually relieves the stiffness and aching; however, prolonged physical activity commonly increases the discomfort. Heat relieves; massage gradually made rather "firm," is usually helpful. Hot packs are helpful for severe localized pain and warm baths are the best means of heating the whole body. Analgesia can be accomplished best by regularly timed doses of acetyl

salicylic acid or one of its derivatives, or by mixtures of non-narcotic analgesics of simple nature.

When trigger points are located, injection with procaine may relieve by breaking the reflex responsible for referred pain. Such injections may be wisely employed several times at three to seven day intervals. Because of the stiffness which characterizes this syndrome, anti-spasmodic drugs such as d-tubocurarine, and 3 ortho-toloxy – 1, 2 – propane-diol (Tolserol) have been tried. Except in acutely painful neck or low back syndromes where muscle spasm is prominent, these anti-spasmodics are of little or no value for the stiffness of fibrositis usually is not due to muscle spasm, as once commonly believed. Roentgen therapy is usually disappointing. "Fibrositic" vaccines, once quite popular, are of no recognizable value and are seldom used. Vitamin E has been recommended for fibrositis and some authors[4] report brilliant results even when used topically.[5] The majority of investigators have found Vitamin E "speculative" or of purely psycho-therapeutic value for fibrositis. Many trials by the author have resulted in failures. Gold salts usually do not help, and are not recommended. Patients with fibrositis should avoid exposure to dampness and cold. Some patients are relieved when in a warm, dry climate.

Because of the noted success in relief of *secondary* fibrositis of rheumatoid arthritis by cortisone or ACTH, it has been considered likely that primary fibrositis would respond readily to these anti-rheumatic hormones. However, prolonged scarcity of these new hormones have prevented sufficient study to clearly define their value in fibrositis. Two patients in our research group responded promptly and completely while doses of cortisone comparable to the average amount used for rheumatoid arthritis were employed for ten to fourteen days but relapse occurred promptly when the hormone was discontinued. A third patient had only about 50 per cent relief from cortisone. Further studies are needed to clarify the position these hormones may have in the management of fibrositis. At present it appears that the dosage requirement will be comparable to that for rheumatoid arthritis and that short use is insufficient. Realizing the mild nature of this disease and the problems associated with the use of these potent hormones, it seems unlikely that their use for fibrositis will prove wise.

Prognosis: Joint deformities and crippling never result from fibrositis. The disease usually subsides or can be controlled by treatment,

although some stubborn cases cause much discomfort for long periods of time. It is important that patients be informed that they have only a mild, "nuisance disease" for they usually fear that they have arthritis which will cripple and incapacitate them if it persists; they are greatly relieved to learn the disease only causes discomfort and temporary difficulty in joint movements. Persistence in a conservative, intelligently planned program of treatment usually gives good results.

Localized and Special Forms of Fibrositis

Fibrous tissue irritation may be localized. The symptoms and dysfunction depend upon the anatomic localization, nature and severity of the abnormalities. Examples of localized fibrositis are the acute, painful, stiff neck or low back (lumbago). Each of these painful, sometimes temporarily incapacitating disorders results from irritation of muscle sheaths, fascia, tendons and ligaments in a portion of the back. Muscle spasm is sometimes severe, hence, anti-spasmodics are more often helpful in this form of fibrositis. Hot fomentations and injections of procaine at the site of the irritation may effect prompt relief. For such severely painful disorders use of codeine or demoral is sometimes justified. Generally, the more acute the onset, and more rapid the development, the more complete and rapid is the resolution of localized fibrositis.

Bursitis: Inflammation of a bursa is a common form of localized fibrositis. Usually the cause cannot be ascertained; occasionally it seems quite clear that the bursitis has been caused by trauma, unusual activity, exposure to dampness or cold, and sometimes by infection. Since bursae exist about all large joints and some small ones, bursitis may occur in many locations. The subacromial, olecranon, ischial, prepatellar and achilles bursae are more commonly affected (Fig. 1). Different from the nature of generalized fibrositis, bursitis is truly an inflammatory process.

One of the most difficult problems of non-articular rheumatism is the "painful shoulder." Fibrositis is much more commonly the cause of shoulder rheumatism than is arthritis. There has been much confusion surrounding the subject of "painful shoulder." To illustrate: Different authors have discussed painful shoulder under the titles bursitis, tendinitis, calcific tendonitis, non-calcific tendonitis, tear of the musculotendinous cuff, "frozen shoulder," periarthritis of shoulder, peritendinous adhesions, adhesive capsulitis, adhesive or obliterative subdeltoid bursitis, calcific bursitis, interstitial fibrosis and occasionally, simply, fibrositis

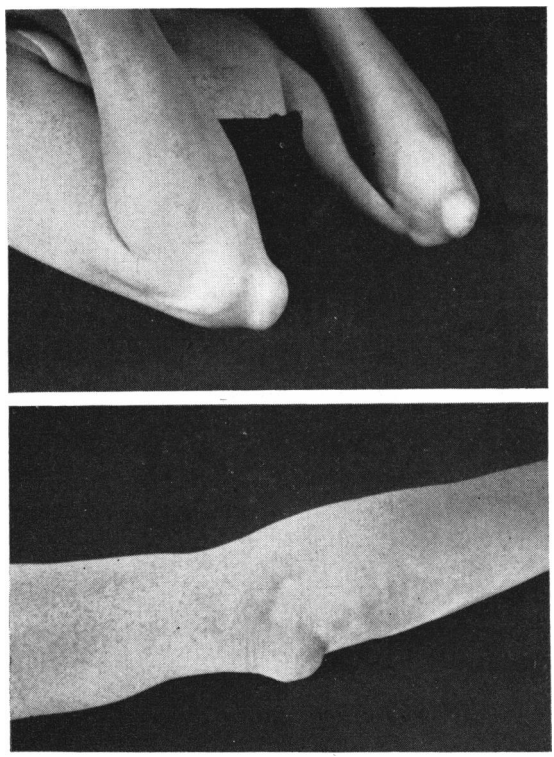

Fig. 1—Characteristic appearance of acute olecranon bursitis with fluid in the bursal space.

of the shoulder. Some of these conditions can be differentiated with accuracy, sometimes they cannot be; then particularly a general understanding of the problem is of paramount importance.

Bursitis about the shoulder may begin precipitously or insidiously and may be acute or chronic with respect to severity and duration. Acute subacromial bursitis (the commonest form of bursitis about the shoulder) may be mild or severe. It is characterized by pain and tenderness localized to subacromial region although it may be more diffuse, and intensified near the humeral insertion of the deltoid. The discomfort is made worse when the arm is hanging unsupported, by pressure over the tip of the shoulder or by movement, especially in abduction and external rotation. Many times these motions are markedly restricted by pain. Fluid may distend the bursal sac and thus add to the pain. This fluid may contain calcium salt in semi-solid consistency.

Because of the proximity of the structures, it is difficult in some cases to determine whether subacromial bursitis or supraspinatous tendinitis is responsible for the symptoms. Irritation of one may secondarily affect the other adjacent tissue so that both may cause the pain simultaneously. Calcific deposits in the tendon near its insertion into the greater tuberosity of the humerus is common, and rupture into the bursa may occur. Abnormal calcium deposits may be absorbed or withdrawn surgically, or remain and become firm, even bony hard, and add to the irritation of adjacent structures so as to account for a chronic painful shoulder which may be very stubborn in response to all non-surgical treatment.

Diagnosis is usually not difficult when the clinical pattern is appreciated. Roentgenograms are negative except for the evidence of abnormal, soft-tissue calcific deposits when they exist.

Treatment should be directed to relieve pain and to maintain function; reassurance should be given that the disorder is localized and comparatively mild. Radiant heat or diathermy may relieve, but sometimes aggravate the pain; then cold applications usually relieve. Rest of the shoulder is advisable during the acute phase; graded exercises to restore motion should be instituted as soon as pain lessens. If the bursal sac is distended, removal of the exudate by syringe, or simply puncturing the sac and allowing the fluid to escape into the surrounding tissue may relieve greatly. Infiltration of the inflamed tissue with procaine may relieve sufficiently to warrant repeated injections. Procaine block of the superior cervical ganglion or brachial plexus will give temporary relief and allow exercise of the shoulder so as to avoid stiffness. Roentgen therapy wisely used in small amounts helps some cases, especially in the subacute stage, with or without calcific deposits. Extensive roentgen irradiation should be avoided. If firm calcific masses persist, excision of the calcium deposit and liberation of the adhesive disorder may be required.

Proper treatment provided promptly and persistently usually relieves discomfort until the inflammation subsides. If the condition becomes chronic, periarticular fibrositis may result and cause stiffness of the shoulder. Periarthritis may occur at the shoulder without initial bursitis. If stiffness of the shoulder persists, muscles atrophy and periarticular adhesions form, and gleno-humeral motion may be lost, a "frozen shoulder" resulting. Careful manipulation of the shoulder while anes-

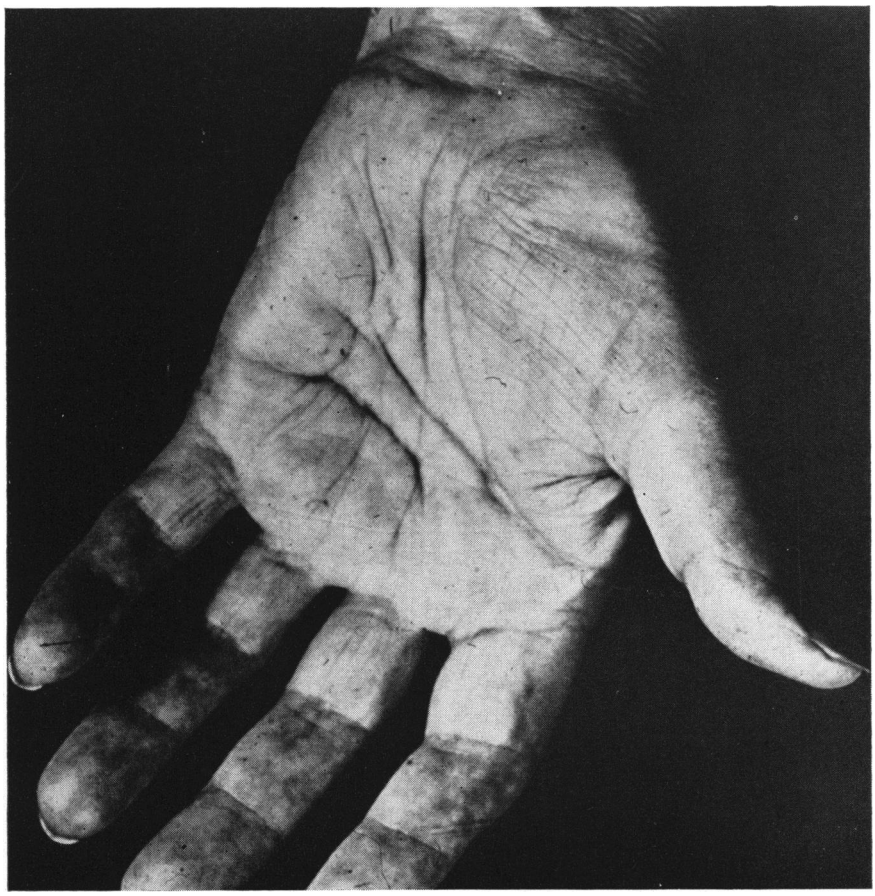

Fig. 2—Palmar fasciitis; note the general flexion deformities of the fingers and the puckering of the palm.

thetized may help to mobilize such a stiff shoulder. Bursitis about other joints produces similar problems; the anatomic localization accounts for the differences in the clinical picture and therapeutic considerations.

Another form of fibrositis involves the tendons primarily. Tendons may become inflamed, although more often they are painful without evidence of inflammation, accounting for the symptom complex known as "tendon attachment syndrome." This may be caused by trauma or by unusual activity and may occur with or without regional bursitis. This difficulty located at the attachment of the conjoined tendon onto the

humeral epicondyle is known as "tennis elbow." Treatment is similar to that for bursitis.

Tenosynovitis: Specific infection, such as tuberculosis may cause inflammation of tendon sheaths; more often cause for tenosynovitis is unknown; sometimes nodular and stenosing changes develop in tendon sheaths so as to impede motion of the encased tendon (DeQuervain's disease). When this condition affects flexor tendon sheaths of a finger, after the finger is flexed, extension may not be possible except with assistance. This condition is called "trigger finger." If warm baths or paraffin dips and exercise do not relieve, incision of the tendon sheaths may be required to free the motion.

Fasciitis and Panniculitis: Generalized inflammation of fascia in the palms of the hands may limit extension of all fingers (Fig. 2). Involvement may be localized to the fascia and tendons to the fourth and fifth digits, producing "Dupuytren's contracture."

Subcutaneous fascia may become inflamed with or without the adjacent fat components resulting in painful nodular subcutaneous swellings. These frequently occur over the abdomen, shoulders and thighs, giving rise to the painful syndrome called "panniculitis."

Herniation of subcutaneous fat through a weak portion of the fascia or a small foramen (recently described as "herniated fat") may occur in the low back and cause pain simulating fibrositis. Copeman and Ackerman[6] dissected the backs of 14 soldiers who died of various causes and found a "basic fat pattern" which coincided with the regions in which what was thought to be "fibrositic pain" and nodules had occurred during life. Instead of subcutaneous ("fibrositic") nodules, small herniations of lobulated fat were found penetrating through fascial tears or through the unprotected foramina by which the posterior primary rami of the first three lumbar nerves pierce the deep fascia. Pain was ascribed to edema in the lobules producing tension which would cause herniation if fascial walls were deficient. At necropsy, fat in the lumbar region could be caused to herniate when pressure was applied to adjacent tissue. Herniations during life were attributed to incidents such as sudden trauma, quick flexion of back, and prolonged rest in the supine position.

Tender subcutaneous "nodules" were removed surgically from patients with back pain and proved to be herniated fat showing no evidence of inflammation. Such herniated fat was believed to be the

"trigger point" of pain in some cases resembling fibrositis. Later cases were treated more conservatively. The overlying skin was anesthetized, the nodule transfixed with a cutting needle from which 10 to 20 cc. procaine solution were injected; then the needle was swept around to undercut the nodule thereby dispersing the edema and tension. Pain and tenderness were reported to be relieved. Since this first report of Copeman others have reported similarly.

Shoulder-Hand Syndrome: This interesting and incompletely understood form of non-articular rheumatism may follow myocardial infarction or other painful intrathoracic lesions, cerebral vascular accidents, irritative lesions in the neck or upper extremity, and sometimes exists without recognizable cause. It is characterized by pain in the shoulder or hand, commonly in both parts; it may be unilateral or bilateral. Shoulder motion becomes limited and the affected hand becomes edematous and painful so that it is held stiff. After several weeks, atrophy occurs in the shoulder and hand and adhesions or contractures limit motion in the affected parts. Severe osteoporosis characterizes the roentgenograms which always show normal joint structure.

This syndrome is thought to be a reflex dystrophy. It is important that it be differentiated from rheumatoid arthritis. Treatment with usual physical measures and analgesics commonly fails. Procaine block of the brachial plexus or superior cervical ganglion is usually an effective means of temporarily allaying pain, which should be accomplished early in the illness, so that functional exercises can be performed to prevent stiffness. If stiffness of shoulder and/or hand results, rehabilitation depends upon persistent physical and occupational therapy.

The effects of cortisone and ACTH are being studied in this syndrome and these hormones may prove valuable at least in some cases.

Psychogenic Rheumatism: A common form of non-articular rheumatism in civilian as well as military practice is "psychogenic rheumatism" —the rheumatic manifestations of psychoneurosis. During World War II it was the commonest form of rheumatism encountered in many hospitals and field areas.[7] Psychoneurotic persons in emotional conflict caused by psychic trauma, fear, anxiety, apprehension or sorrow may experience arthralgia, muscle and tendon pain, stiffness and interference with joint motion resembling common forms of arthritis or fibrositis. The true nature of this condition must be recognized; this is usually not difficult when psychogenic rheumatism exists alone. Absence of joint

changes on clinical and roentgenologic examination, normal laboratory observations, and good organic health are characteristic. Bizarre deformities not conforming to anatomic changes and recognition of other evidence of psychoneurosis are helpful diagnostic features. It may be difficult to differentiate psychogenic rheumatism from fibrositis. In the patient with psychogenic rheumatism commonly symptoms are not localized or consistent. There is lack of benefit from analgesics, and physical therapy and failure to improve in warm atmosphere and after mild exercise—all usually found in patients with fibrositis. Treatment should be directed to relieving emotional conflict and rationalization of the problems. Unless these objectives are realized results will be disappointing.

Summary

About one out of every four persons attending arthritis clinics in the United States does not have arthritis, but has a form of rheumatism affecting only non-articular fibrous connective tissue. Symptoms of non-articular rheumatism are identical with or very similar to those of some forms of arthritis. There are many different clinical varieties of non-articular rheumatism. It is important to differentiate non-articular rheumatism from arthritis, and to ascertain which type of non-articular rheumatism exists, for the proper treatment and prognosis differ.

Usually non-articular rheumatism is only a "nuisance disorder" and proper management gives satisfactory results. The patient should be reassured that the disease is not arthritis, that it is usually mild, and although oftentimes slow to improve, the prognosis is good.

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